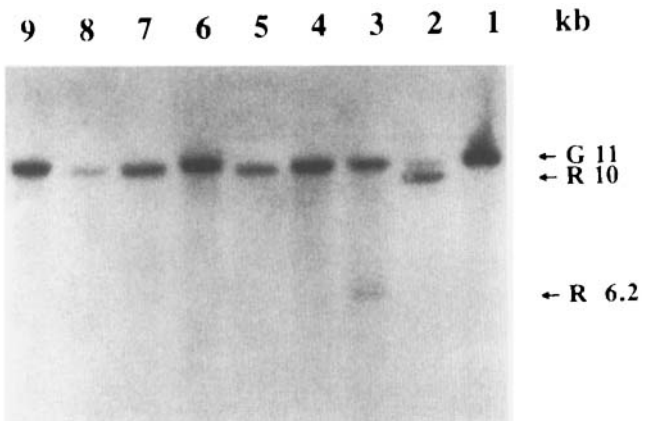

Fig. 1. Southern analysis using a *bcl*-6 probe following *Bam*HI digestion. Lane 1: Normal control. Lane 2: A case of diffuse large B-cell lymphoma with bcl-6 rearrangement. Lane 3: The case of LPHD reported here. Lanes 4–9: Other cases of lymphoma with no *bcl*-6 rearrangement.

by R. Dalla-Favera of Columbia University was used as probe [1]. Following *Bam*HI digestion, a rearranged band of 6.2 kb was found in addition to the 11-kb germline band (lane 3, Fig. 1). Also, following *xba*I digestion, a 5.6-kb rearranged band was seen in addition to the 13.5-kb germline band. They confirmed the presence of *bcl*-6 rearrangement in this case.

Many investigators have demonstrated B-cell-associated molecules in the L&H cells of LPHD. The tumor is considered to be of B-cell origin or to represent a large B-cell lymphoma in evolution [2–5]. Attempts have been made to demonstrate the clonality of LPHD. Techniques employed include Southern blot analysis and polymerase chain reaction (PCR) for detection of clonal immunoglobulin gene (Ig) rearrangement [2]. In situ analysis of immunoglobulin heavy chain protein or mRNA expression has also been used. Unfortunately, these methods have yielded conflicting results [5]. A new technique that isolated a single L&H cell for PCR detection of Ig gene heavy chain rearrangement has provided evidence for a B-cell origin but a polyclonal nature of the L&H cells [3].

This reported case has provided new insight into the biology of LPHD. The detection of a *bcl*-6 rearrangement in LPHD strongly suggests a clonal nature for LPHD and an intimate relationship between the tumor and non-Hodgkin's lymphoma of the diffuse large B-cell type. Further studies are necessary to define the incidence of *bcl*-6 gene rearrangement in LPHD and the precise relationship with large B-cell lymphoma.

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Two Cases of Epidemic Mucormycosis Infection in Patients With Acute Lymphoblastic Leukemia

To the Editor: Infection caused by fungus of the Mucorales order is uncommon and has been described almost exclusively in leukopenic patients who are undergoing chemotherapy [1]. We observed this rare opportunistic infection in two patients treated for acute lymphoblastic leukemia (ALL) at our institution. The first was a 40-year-old man with ALL2 who relapsed 11 months after completion of treatment. During the consolidation course of chemotherapy, febrile pneumopathy developed and bronchoalveolar lavage (BAL) identified *Xanthomonas maltophilia* associated with mucormycosis. Despite effective antibiotherapy against *Xanthomonas* and treatment with amphotericin B (Amb), the patient died of massive hemoptysis. Necropsy revealed rupture of the right auricle in the main right bronchus.

The second patient was a 35-year-old woman with a Ph1+ B ALL. During the aplasia induced by the consolidation treatment with high-dose cytosine-arabinoside plus amsacrine, the diagnosis of pulmonary filamentous mycosis was made on a BAL, and mucormycosis was identified on a liver biopsy done for two abscesses seen on echography. Stabilization of the hepatic lesion and 50% reduction of the pneumopathy was obtained by AmB and liposomal AmB. Three months later, while still in complete cytogenetic and molecular remission, she underwent pulmonary lobectomy and hepatic segmentectomy. Hepatic mucormycosis was confirmed, and mucormycosis was found associated with aspergillosis in the lung. Allogeneic bone marrow transplantation (BMT) was performed 2 months later for cytologic relapse. She died of extensive aspergillosis 90 day after BMT.

Mucormycosis is a rare fungal infection whose prognosis is almost always fatal in patients with acute leukemia [2]. One portal entry is the respiratory tract. The fungus can cause thrombosis, ischemia, and hemorrhage by erosion of the blood vessels. Early antimycotic therapy seems essential to improve the prognosis, but surgical resection of the infected tissue may be life saving.

Our two cases illustrate the risk of fatal hemoptysis associated with mucormycosis infection and the benefit of surgical resection recently reported by Pagano et al. [3]. Moreover, these cases point out several facts: 1) the two patients had received high-dose corticosteroid therapy, which has been shown to enhance *Aspergillus* growth in vitro [4]; the association of *Mucor* and aspergillosis suggests that the same risk factors, (corticosteroids and immunosuppression) are required for both; 2) even though surgical treatment of mucor abscesses was efficient in the second patient, aspergillosis developed despite AmB, liposomal AmB, and itraconazole therapy after BMT; the feasibility of BMT in patients with apparently cured aspergillosis is thus in question [5]; and 3) the major point is the epidemic risk of mucormycosis infection. These two patients stayed successively in the same room during aplasia; systematic control of surfaces led us to discover *Mucor* organisms on the ventilation grating. To our knowledge, this is the first

case of epidemic mucormycosis; we stress the necessity to control all surfaces of a room in which a patient has been infected.

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Translocation 21;22 May Be Involved in Control of Differentiation in Erythroleukemia

To the Editor: A 61-year-old man was admitted to Urafune Hospital of Yokohama City University with severe pancytopenia in September, 1994. The hemoglobin was 7.3 g/dl, white blood cell (WBC) count 1,000/μl with 87% lymphocytes, platelet count 25,000/μl, and erythroblast count 4/100 WBC. Bone marrow examination showed 52.4% erythroblasts, 37.2% myeloblasts of all nucleated cells, and 92.1% myeloblasts of nonerythroid cells. The blasts were negative to periodic acid-Schiff stain and α-naphtylbutylate esterase and positive to peroxidase. Morphology study showed that the blasts had large azurophilic granules, an atypical nucleus, and frequently Auer rods [Fig. 1]. Many erythroblasts showed megaloblastoid changes. Immunophenotypic analysis of the blasts revealed a positive reaction for CD13, CD33, and HLA-DR. Cytogenetic analysis was performed on bone marrow cells using G banding. All of 16 metaphases revealed a 21;22 translocation: 46,XY,t(21;22)(22q;q1?). On the basis of these results, we diagnosed acute non-lymphocytic leukemia (FAB-M6) and treated the patient with daunorbicin, cytosine arabinoside, 6-mercaptopurine, and prednisolone. The patient died of sepsis in October, 1994. We tried to examine bone marrow mononuclear cells (cryoreserved on admission) with regard to AML1 and major *bcr/abl* fusion genes by fluorescence in situ hybridization (FISH), but these genes could not be detected.

Olopade et al. [1] reported that erythroleukemia showed chromosomal abnormalities in over 70% of cases and generally clonal abnormalities led to loss of all or part of the long arm of chromosome 5 and/or chromosome 7. Chromosomal abnormalities on a 21;22 translocation have been previously reported in three cases of chronic myelogenous leukemia [2–4] but have not been noted in acute leukemia. One of the translocation in M2 is the t(8;21)(q22;q22), which has recently been shown to involve the AML1 gene at 21q22, and it has been suggested that the AML1 gene may be involved in control of cellular proliferation and/or differentiation [5]. Although we could not detect AML1 and major *bcr/abl* fusion genes by FISH, it was thought that in this case a 21;22 translocation caused differentiation in leukemic cells with azurophilic granules.

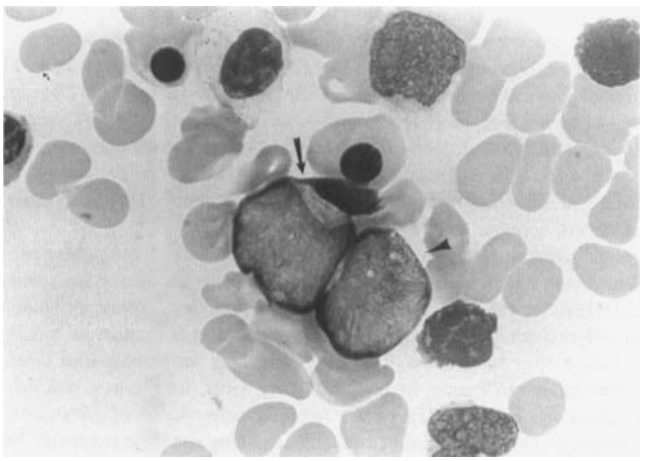

Fig. 1. Blast cells in bone marrow, with Auer rods (arrow) and azurophilic granules (arrowhead). (Wright-Giemsa stain, ×1,000.)

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Hodgkin's Lymphoma in a Cyclist Treated With Growth Hormone

To the Editor: The misuse of growth hormone (GH) to enhance athletic performance may become a significant challenge to the sports world. It is apparent that doping is increasingly used also by amateur and non-competi-